WEST Search History

DATE: Friday, May 02, 2003

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=AND			
L7	andrew.in. and yoo.in.	2	L7
L6	l1 and L5	0	L6
L5	rudolph.in. and tanzi.in.	14	L5
L4	tae-wan.in. and kim.in.	2	L4
L3	l1 and L2	12	L3
L2	alzheimers	9706	L2
L1	capacitative adj calcium adj entry	19	L1

END OF SEARCH HISTORY

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FILE 'HOME' ENTERED AT 18:30:56 ON 02 MAY 2003

=> file biosis caplus medline

COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'BIOSIS' ENTERED AT 18:31:41 ON 02 MAY 2003
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FILE 'MEDLINE' ENTERED AT 18:31:41 ON 02 MAY 2003

=> capacitative calcium entry

L1          640 CAPACITATIVE CALCIUM ENTRY

=> cce

L2         1684 CCE

=> l2 and calcium

L3          352 L2 AND CALCIUM

=> l1 or l3

L4          848 L1 OR L3

=> l4 and method?

L5           92 L4 AND METHOD?

=> l5 and 1970-2000/py

L6           36 L5 AND 1970-2000/PY

=> dup rem l6

PROCESSING COMPLETED FOR L6
L7           29 DUP REM L6 (7 DUPLICATES REMOVED)

=> l1 and alzheimers

L8            0 L1 AND ALZHEIMERS

=> l2 and alzheimers

L9            0 L2 AND ALZHEIMERS

=> l1 and alzheimer?

L10          17 L1 AND ALZHEIMER?
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=> l10 and 1970-2000/py

L11 13 L10 AND 1970-2000/PY

=> dup rem l11

PROCESSING COMPLETED FOR L11
L12 8 DUP REM L11 (5 DUPLICATES REMOVED)

=> d ti abs so l12 1-8

L12 ANSWER 1 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
TI Presenilins, **Alzheimer**'s disease, and **capacitative calcium entry**.
SO Neuron, (**September, 2000**) Vol. 27, No. 3, pp. 411-412. print.
ISSN: 0896-6273.

L12 ANSWER 2 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
TI **Capacitative calcium entry** deficits and elevated luminal calcium content in mutant presenilin-1 knockin mice.
AB Dysregulation of calcium signaling has been causally implicated in brain aging and **Alzheimer**'s disease. Mutations in the presenilin genes (PS1, PS2), the leading cause of autosomal dominant familial **Alzheimer**'s disease (FAD), cause highly specific alterations in intracellular calcium signaling pathways that may contribute to the neurodegenerative and pathological lesions of the disease. To elucidate the cellular mechanisms underlying these disturbances, we studied calcium signaling in fibroblasts isolated from mutant PS1 knockin mice. Mutant PS1 knockin cells exhibited a marked potentiation in the amplitude of calcium transients evoked by agonist stimulation. These cells also showed significant impairments in **capacitative calcium entry** (CCE, also known as store-operated calcium entry), an important cellular signaling pathway wherein depletion of intracellular calcium stores triggers influx of extracellular calcium into the cytosol. Notably, deficits in CCE were evident after agonist stimulation, but not if intracellular calcium stores were completely depleted with thapsigargin. Treatment with ionomycin and thapsigargin revealed that calcium levels within the ER were significantly increased in mutant PS1 knockin cells. Collectively, our findings suggest that the overfilling of calcium stores represents the fundamental cellular defect underlying the alterations in calcium signaling conferred by presenilin mutations.
SO Journal of Cell Biology, (**May 15, 2000**) Vol. 149, No. 4, pp. 793-797. print.
ISSN: 0021-9525.

L12 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
TI Presenilin-mediated modulation of **capacitative calcium entry**.
AB We studied a novel function of the presenilins (PS1 and PS2) in governing **capacitative calcium entry** (CCE), a refilling mechanism for depleted intracellular calcium stores. Abrogation of functional PS1, by either knocking out PS1 or expressing inactive PS1, markedly potentiated CCE, suggesting a role for PS1 in the modulation of CCE. In contrast, familial **Alzheimer**'s disease (FAD)-linked mutant PS1 or PS2 significantly attenuated CCE and store depletion-activated currents. While inhibition of CCE selectively

increased the amyloidogenic amyloid BETA peptide (ABETA42), increased accumulation of the peptide had no effect on CCE. Thus, reduced CCE is most likely an early cellular event leading to increased ABETA42 generation associated with FAD mutant presenilins. Our data indicate that the CCE pathway is a novel therapeutic target for **Alzheimer**'s disease.

SO Neuron, (**September, 2000**) Vol. 27, No. 3, pp. 561-572. print. ISSN: 0896-6273.

L12 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2003 ACS

TI Presenilins, **Alzheimer**'s disease, and **capacitative calcium entry**

AB The title research of A.S. Yoo, et al. (ibid.) is reviewed with commentary and 13 refs.

SO Neuron (**2000**), 27(3), 411-412 CODEN: NERNET; ISSN: 0896-6273

L12 ANSWER 5 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

TI Calcium signaling alterations due to mutations in presenilin-1 and -2.

AB Mutations in the presenilin-1 and -2 (PS1, PS2) genes cause early-onset familial **Alzheimer**'s disease (FAD), but the precise pathogenic mechanisms remain to be established. In addition to modulating beta-amyloid production, FAD mutations in both PS1 and PS2 consistently produce highly specific and selective changes to intracellular calcium signaling pathways. Specifically, we have shown that presenilin mutations enhance phosphoinositide-mediated calcium release from intracellular stores and impair **capacitative calcium entry** . To elucidate the subcellular mechanisms underlying these changes, we used high-resolution confocal line-scanning microscopy to study elementary calcium release events in Xenopus oocytes expressing wild type or mutant PS1. We found that PS1 mutations increase the total amount of calcium released per elementary event through a mechanism involving increased rate of calcium flux. Studies in mutant PS1 knock-in mice suggest that an overfilling of intracellular calcium stores represents the fundamental cellular defect underlying the perturbations in calcium signaling. We will also present data suggesting a potential role for gamma-secretase activity in the modulation of intracellular calcium signaling pathways.

SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-492.3. print. Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience . ISSN: 0190-5295.

L12 ANSWER 6 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

TI Altered calcium signaling in cells lacking presenilin-associated gamma-secretase activity.

AB Mutations in the presenilin genes (PS1, PS2) are a leading cause of early-onset familial **Alzheimer**'s disease (FAD). Two highly consistent consequences of presenilin mutations are: (1) increased gamma-secretase cleavage of the beta-amyloid precursor protein (APP) and (2) specific alterations of intracellular calcium signaling pathways, including enhanced calcium release from intracellular stores and deficits in **capacitative calcium entry** (Leissring et al., J. Cell Biol. 149, in press). To investigate the relationship between gamma-secretase activity and calcium dysregulation, we are studying

calcium signaling in cell lines stably transfected with APP together with PS1 and PS2 constructs containing mutations in critical aspartyl residues required for gamma-secretase activity. Relative to untransfected cells, cells expressing APP alone exhibited a significant potentiation in calcium signals evoked by agonists coupled to the phosphoinositide signaling cascade. Calcium signals in cells co-expressing APP together with PS1 and PS2 aspartyl mutations were potentiated to a significantly greater extent and also showed alterations consistent with enhanced **capacitative calcium entry**. Comparable studies using fibroblasts from PS1 and PS2 knock-out mice are in progress. These results will establish the relationship between the gamma-secretase activity in the modulation of intracellular calcium signaling pathways.

SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-474.7. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience . ISSN: 0190-5295.

L12 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

TI Presenilin-mediated modulation of calcium release-activated calcium current (ICRAC)and the Abeta42 biogenesis.

AB Perturbed Ca2+ homeostasis is one of the characteristic molecular phenotypes associated with presenilin familial **Alzheimer**'s disease (FAD) mutations. We have previously reported that the capacitative Ca2+ entry (CCE) response, which is triggered by an intracellular Ca2+ store depletion, is significantly attenuated in cells expressing FAD mutant presenilins as compared to wild-type presenilins. To begin to elucidate the underlying mechanism for reduced CCE in presenilin FAD mutant cells, we studied whether FAD-associated PS1 mutation directly affect the activity of Ca2+ release-activated Ca2+ channels (ICRAC), which is a Ca2+-specific putative plasma membrane channel. ICRAC channel activities were measured by whole-cell mode patch clamp technique using CHO or SY5Y cell lines stably expressing either wild-type or M146L FAD mutant forms of PS1. We found that ICRAC were severely impaired in the cells expressing mutant PS1 as compared to wild-type PS1, indicating that aberrant ICRAC may be responsible for attenuated CCE in cells harboring FAD mutant presenilins. We have also previously demonstrated that CCE pathway is directly coupled to the presenilin-dependent gamma-secretase activity. We next tested whether augmentation of CCE would directly modulate the biogenesis of Abeta42. For this purpose, we ectopically expressed constructs encoding three different isoforms of putative store-operated Ca2+ channels, termed TRP (e.g. TRP1, TRP3, and TRP6; gifts of Drs. Craig Montell and Lutz Birnbaumer). TRP3 and TRP6 overexpression potentiates CCE in cells expressing FAD mutant presenilins. Interestingly, the generation of Abeta42 was decreased by TRP-transfection as compared to untransfected M146L-PS1 cells. Thus, augmentation of CCE (through TRP or relevant cellular components) could potentially be employed to reduce presenilin-associated gamma-secretase activity, and therefore Abeta42 generation.

SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-474.9. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience . ISSN: 0190-5295.

L12 ANSWER 8 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI The N141I presenilin 2 familial **Alzheimer** mutation is associated with abated proteasomal degradation and reduced **capacitative calcium entry**.
SO Society for Neuroscience Abstracts, (1999) Vol. 25, No. 1-2, pp. 1568. Meeting Info.: 29th Annual Meeting of the Society for Neuroscience. Miami Beach, Florida, USA October 23-28, 1999 Society for Neuroscience . ISSN: 0190-5295.

=> andrew?/au and yoo?/au

L13 2 ANDREW?/AU AND YOO?/AU

=> d ti abs so l13 1-2

L13 ANSWER 1 OF 2 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI OPTIMIZATION OF THE STANDING VERTICAL JUMP.
SO 2ND ANNUAL MEETING OF THE AMERICAN SOCIETY OF BIOMECHANICS, ANN ARBOR, MICH., USA, OCT. 26-27, 1978. J BIOMECH. (1979) 12 (8), 637. CODEN: JBMCB5. ISSN: 0021-9290.

L13 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
TI Experimental study on heat transfer in two-phase loop thermosyphon for cooling of MCM
AB The thermal performance of two-phase loop thermosiphon systems designed for the ISDN telecommunications system has been investigated. The loop thermosiphon design was proposed to cool a multichip module (MCM) simulated for a max. heat dissipation of 5 W/cm2. As a design tool, the thermal resistance was considered. The temp. rise and its dimensionless distributions were compared for different cooling conditions and heat densities of the evaporator section. It was found that the two phase loop thermosiphon performed well having a temp. of 75 .degree.C at the evaporator under the high thermal load of 5 W/cm2.
SO Heat Pipe Technology: Theory, Applications and Prospects, Proceedings of the International Heat Pipe Symposium, 5th, Melbourne, Nov. 17-20, 1996 (1997), 223-229. Editor(s): **Andrews, J.; Akbarzadeh, A.; Sauciuc, I.** Publisher: Elsevier, Oxford, UK. CODEN: 66GKA8

=> rudolph?/au and tanzi?/au

L14 0 RUDOLPH?/AU AND TANZI?/AU

=> tae-wan?/au and kim?/au

L15 0 TAE-WAN?/AU AND KIM?/AU

=> l1 and neurodegener?

L16 6 L1 AND NEURODEGENER?

=> l16 and 1970-2000/py

L17 3 L16 AND 1970-2000/PY

=> dup rem l17

PROCESSING COMPLETED FOR L17
L18 1 DUP REM L17 (2 DUPLICATES REMOVED)

=> d ti abs so l18

L18 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1

TI **Capacitative calcium entry** deficits and elevated luminal calcium content in mutant presenilin-1 knockin mice.

AB Dysregulation of calcium signaling has been causally implicated in brain aging and Alzheimer's disease. Mutations in the presenilin genes (PS1, PS2), the leading cause of autosomal dominant familial Alzheimer's disease (FAD), cause highly specific alterations in intracellular calcium signaling pathways that may contribute to the **neurodegenerative** and pathological lesions of the disease. To elucidate the cellular mechanisms underlying these disturbances, we studied calcium signaling in fibroblasts isolated from mutant PS1 knockin mice. Mutant PS1 knockin cells exhibited a marked potentiation in the amplitude of calcium transients evoked by agonist stimulation. These cells also showed significant impairments in **capacitative calcium entry** (CCE, also known as store-operated calcium entry), an important cellular signaling pathway wherein depletion of intracellular calcium stores triggers influx of extracellular calcium into the cytosol. Notably, deficits in CCE were evident after agonist stimulation, but not if intracellular calcium stores were completely depleted with thapsigargin. Treatment with ionomycin and thapsigargin revealed that calcium levels within the ER were significantly increased in mutant PS1 knockin cells. Collectively, our findings suggest that the overfilling of calcium stores represents the fundamental cellular defect underlying the alterations in calcium signaling conferred by presenilin mutations.

SO Journal of Cell Biology, (**May 15, 2000**) Vol. 149, No. 4, pp. 793-797. print.
ISSN: 0021-9525.

=> d his

(FILE 'HOME' ENTERED AT 18:30:56 ON 02 MAY 2003)

FILE 'BIOSIS, CAPLUS, MEDLINE' ENTERED AT 18:31:41 ON 02 MAY 2003

L1	640	CAPACITATIVE CALCIUM ENTRY
L2	1684	CCE
L3	352	L2 AND CALCIUM
L4	848	L1 OR L3
L5	92	L4 AND METHOD?
L6	36	L5 AND 1970-2000/PY
L7	29	DUP REM L6 (7 DUPLICATES REMOVED)
L8	0	L1 AND ALZHEIMERS
L9	0	L2 AND ALZHEIMERS
L10	17	L1 AND ALZHEIMER?
L11	13	L10 AND 1970-2000/PY
L12	8	DUP REM L11 (5 DUPLICATES REMOVED)
L13	2	ANDREW?/AU AND YOO?/AU
L14	0	RUDOLPH?/AU AND TANZI?/AU
L15	0	TAE-WAN?/AU AND KIM?/AU
L16	6	L1 AND NEURODEGENER?
L17	3	L16 AND 1970-2000/PY
L18	1	DUP REM L17 (2 DUPLICATES REMOVED)

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